SECTION 4

Note: Some figures and tables are not available on the Internet version.

TARGET ANALYTES

The selection of appropriate target analytes in fish and shellfish contaminant monitoring programs is essential to the adequate protection of the health of fish and shellfish consumers. The procedures used for selecting target analytes for screening studies and a list of recommended target analytes are presented in this section.

4.1 RECOMMENDED TARGET ANALYTES

Recommended target analytes for screening studies in fish and shellfish contaminant monitoring programs are listed in Table 4-1. This list was developed by the EPA Fish Contaminant Workgroup from a review of the following information:

Table 4-1. Recommended Target Analytes [a]

Metals
- Arsenic (inorganic)
- Cadmium
- Mercury
- Selenium
- Tributyltin

Organochlorine Pesticides
- Chlordane, total (cis- and trans-chlordane, cis- and trans-nonachlor, oxychlordane)
- DDT, total (2,4'-DDD, 4,4'-DDD, 2,4'-DDE, 4,4'-DDE, 2,4'-DDT, 4,4'-DDT)
- Dicofol
- Dieldrin
- Endosulfan (I and II)
- Endrin
- Heptachlor epoxide [b]
- Hexachlorobenzene
- Lindane (γ-hexachlorocyclohexane; γ-HCH)[c]
- Mirex[d]
- Toxaphene

Organophosphate Pesticides[e]
- Chlorpyrifos
- Diazinon
- Disulfoton
- Ethion
- Terbufos

Chlorophenoxy Herbicides
- Oxyfluorfen

PAHs[f]

PCBs
- Total Aroclors[g]

Dioxins/furans[h,i]

PAHs = Polycyclic aromatic hydrocarbons.
PCBs = Polychlorinated biphenyls.

[a] States should include all recommended target analytes in screening studies, if resources allow, unless historic tissue or sediment data indicate that an analyte is not present at a level of concern for human health. Additional target analytes should be included in screening studies if States have site-specific information (e.g., historic tissue or sediment data, discharge monitoring reports from municipal and industrial sources, pesticide use application information) that these chemicals may be present at levels of concern for human health.

[b] Heptachlor epoxide is not a pesticide but is a metabolite of the pesticide heptachlor.

[c] Also known as γ-benzene hexachloride (γ-BHC).

[d] Mirex should be regarded primarily as a regional target analyte in the southeast and Great Lakes States, unless historic tissue, sediment, or discharge data indicate the likelihood of its presence in other areas.

[e] The reader should note that carbophenothion was included on the original list of target analytes. Because the registrant did not support reregistration of this chemical, it will no longer be used. For this reason and because of its use profile, carbophenothion was removed from the recommended list of target analytes.

[f] It is recommended that, in both screening and intensive studies, tissue samples be analyzed for benzo[*a*]pyrene, benz[*a*]-anthracene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, chrysene, dibenz[*a,h*]anthracene, and indeno *[1,2,3-cd*]pyrene, and that the order-of-magnitude relative potencies given for these PAHs in the EPA provisional guidance for quantitative risk assessment of PAHs (U.S. EPA, 1993c) be used to calculate a potency equivalency concentration (PEC) for each sample for comparison with the recommended SV for benzo[*a*]pyrene (see Section 5.3.2.3). At this time, EPA's recommendation for risk assessment of PAHs (U.S. EPA, 1993c) is considered provisional because quantitative risk assessment data are not available for all PAHs. This approach is under Agency review and over the next year will be evaluated as new health effects benchmark values are developed. Therefore, the method provided in this guidance document is subject to change pending results of the Agency's reevaluation.

[g] Analysis of total PCBs, as the sum of Aroclor equivalents, is recommended in both screening and intensive studies because of the lack of adequate toxicologic data to develop screening values (SVs) for individual PCB congeners (see Section 4.3.5). However, because of the wide range of toxicities among different PCB congeners and the effects of metabolism and degradation on Aroclor composition in the environment, congener analysis is deemed to be a more scientifically sound and accurate method for determining total PCB concentrations. Consequently, States that currently do congener-specific PCB analyses should continue to do so. Other States are encouraged to develop the capability to conduct PCB congener analysis.

[h] Note: The EPA Office of Research and Development is currently reassessing the human health effects of dioxins/furans.

[i] Dioxins/furans should be considered for analysis primarily at sites of pulp and paper mills using a chlorine bleaching process and at industrial sites where the following organic compounds are formulated: herbicides (containing 2,4,5-trichlorophenoxy acids and 2,4,5-trichlorophenol), hexachlorophene, pentachlorophenol, and PCBs (U.S. EPA, 1987d). It is recommended that the 2,3,7,8-substituted tetra- through octa-chlorinated dibenzo-p-dioxins (PCDDs) and dibenzofurans (PCDFs) be determined and a toxicity-weighted total concentration calculated for each sample (Barnes and Bellin, 1989; U.S. EPA, 1987d) (see Section 5.3.2.4). If resources are limited, 2,3,7,8-TCDD and 2,3,7,8-TCDF should be determined at a minimum.

1. **Pollutants analyzed in several national or regional fish contaminant monitoring programs—** The monitoring programs reviewed included

   - National Study of Chemical Residues in Fish (U.S. EPA)
   - National Dioxin Study (U.S. EPA)
   - 301(h) Monitoring Program (U.S. EPA)
   - National Pollutant Discharge Elimination System (U.S. EPA)
   - National Pesticide Monitoring Program (U.S. FWS)
   - National Contaminant Biomonitoring Program (U.S. FWS)
   - National Status and Trends Program (NOAA)
   - Great Lakes Sportfish Consumption Advisory Program
   - FDA recommendations
   - National Water-Quality Assessment Program (USGS).

   Criteria for selection of the target analytes in these programs varied widely depending on specific program objectives. The target analytes used in these major fish contaminant monitoring programs are compared in Appendix C. Over 200 potential contaminants are listed, including metals, pesticides, base/neutral organic compounds, dioxins, dibenzofurans, acidic organic compounds, and volatile organic compounds.

2. **Pesticides with active registrations—** The EPA Office of Pesticide Programs (OPPs) Fate One Liners Database (U.S. EPA, 1993a) containing information for more than 900 registered pesticides was reviewed to identify pesticides and herbicides with active registrations that met four criteria. The screening criteria used were:

   - Oral toxicity, Class I or II
   - Bioconcentration factor greater than 300
   - Half-life value of 30 days or more
   - Initial use application profile.

   At the time of this review, complete environmental fate information was available for only about half of the registered pesticides. As more data become available, additional pesticides will be evaluated for possible inclusion on the target analyte list.

   Use of the OPP Database was necessary because many pesticides and herbicides with active registrations have not been monitored extensively either in national or State fish contaminant monitoring programs.

3. **Contaminants that have triggered States to issue fish and shellfish consumption advisories or bans—** The database, *National Listing of State Fish and Shellfish Consumption Advisories and Bans* (RTI, 1993), was reviewed to identify specific chemical contaminants that have triggered issuance of consumption advisories by the States. As shown in Table 4-2, four contaminants have triggered advisories in the largest number of States: polychlorinated biphenyls (PCBs), mercury, chlordane, and dioxins/furans.

Table 4-2. Contaminants Resulting in Fish and Shellfish Advisories

Contaminant	Number of States issuing advisories
Metals	
Arsenic (total)	1
Cadmium	2
Chromium	1
Copper	1
Lead	4
Mercury	27
Selenium	5
Zinc	1
Organometallics	1
Unidentified metals	3
Pesticides	
Chlordane	24
DDT and metabolites	9
Dieldrin	3
Heptachlor epoxide	1
Hexachlorobenzene	2
Kepone	1
Mirex	3
Photomirex	1
Toxaphene	2
Unidentified pesticides	2
Polycyclic aromatic hydrocarbons (PAHs)	3
Polychlorinated biphenyls (PCBs)	31
Dioxins/furans	22
Other chlorinated organics	
Dichlorobenzene	1
Hexachlorobutadiene	1
Pentachlorobenzene	1
Pentachlorophenol	1
Tetrachlorobenzene	2
Tetrachloroethane	1
Others	
Creosote	2
Gasoline	1
Multiple pollutants	2
Phthalate esters	1
Polybrominated biphenyls (PBBs)	1
Unspecified pollutants	3

Source: RTI, 1993.

4. **Published literature on the chemistry and health effects of potential contaminants—** The physical, chemical, and toxicologic factors considered to be of particular importance in developing the recommended target analyte list were

   - Oral toxicity
   - Potential of the analyte to bioaccumulate
   - Prevalence and persistence of the analyte in the environment
   - Biochemical fate of the analyte in fish and shellfish
   - Human health risk of exposure to the analyte via consumption of contaminated fish and shellfish
   - Analytical feasibility.

Final selection of contaminants for the recommended target analyte list (Table 4-1) was based on their frequency of inclusion in national monitoring programs, on the number of States issuing consumption advisories for them, and on their origins, chemistry, potential to bioaccumulate, estimated human health risk, and feasibility of analysis. Primary consideration was given to the recommendations of the Committee on Evaluation of the Safety of Fishery Products, published in *Seafood Safety* (NAS, 1991), and to the recommendations of the EPA Fish Contaminant Workgroup.

4.2 SELECTION OF TARGET ANALYTES

States should include all recommended target analytes (Table 4-1) in screening studies, if resources allow, unless historic tissue or sediment or pollutant source data indicate that an analyte is not present at a level of concern (see Section 5). For the pesticides with active registrations, use and rate application information maintained by the State's Department of Agriculture should be reviewed to identify watersheds where these pesticides have been used historically or are currently used and are likely to be present in aquatic systems as a result of agricultural runoff or drift.

It is important to note that pesticide uses and labels may change over time. The State agency responsible for designing the fish contaminant monitoring program should be aware of all historic and current uses of each pesticide within its State, including the locations, application rates, and acreage where the pesticide has been or currently is applied to ensure that all potentially contaminated sites are included in the sampling plan.

Additional target analytes should be included in screening programs if States have site-specific chemical information (e.g., historic tissue or sediment data, discharge monitoring reports from municipal and industrial sources, or pesticide use data) that these contaminants may be present at levels of concern for human health. Compounds that are currently under review by the EPA Office of Water for inclusion as recommended target analytes are discussed in Section 4.4. Specific factors that were considered in the selection of currently recommended target analytes are summarized in the following sections.

4.3 TARGET ANALYTE PROFILES

4.3.1 Metals

Five metals—arsenic, cadmium, mercury, selenium and tributyltin—are recommended as target analytes in screening studies. Arsenic, cadmium, and mercury have been included in six major fish contaminant monitoring programs (see Appendix C). It should be noted, however, that with respect to arsenic, all monitoring programs measured total arsenic rather than inorganic arsenic. Selenium has been monitored in five national monitoring programs. Tributyltin has been recommended for analysis in the FDA monitoring program. Consumption advisories are currently in effect for arsenic, cadmium, mercury, selenium, and tributyltin in one, two, twenty-seven, five, and one States respectively (Table 4-2). Also, with the exception of tributyltin, these metals have been identified as having the greatest potential toxicity resulting from ingestion of contaminated fish and shellfish (NAS, 1991).

4.3.1.1 Arsenic

Arsenic is the twentieth most abundant element in the earth's crust and naturally occurs as a sulfide in a variety of mineral ores containing copper, lead, iron, nickel, cobalt, and other metals (Eisler, 1988; Merck Index, 1989; Woolson, 1975). Arsenic is released naturally to the atmosphere from volcanic eruptions and forest fires (Walsh et al., 1979) and to water via natural weathering processes (U.S. EPA, 1982b). Arsenic also has several major anthropogenic sources including industrial emissions from coal-burning electric generating facilities, releases, as a byproduct of nonferrous-metal (gold, silver, copper, lead, uranium, and zinc) mining and smelting operations (Eisler, 1988; May and McKinney, 1981; NAS, 1977), releases associated with its production and use as a wood preservative (primarily as arsenic trioxide), and application as an insecticide, herbicide, algicide, and growth stimulant for plants and animals (Eisler, 1988). Releases are also associated with leaching at hazardous waste disposal sites and discharges from sewage treatment facilities. Arsenic trioxide is the arsenic compound of chief commercial importance (U.S. EPA, 1982b) and was produced in the United States until 1985 at the ASARCO smelter near Tacoma, Washington. Arsenic is no longer produced commercially within the United States in any significant quantities, but arsenic compounds are imported into the United States primarily for use in various wood preservative and pesticide formulations.

The toxicity of arsenicals is highly dependent upon the nature of the compounds, and particularly upon the valency state of the arsenic atom (Frost, 1967; Penrose, 1974; Vallee et al., 1960). Typically, compounds containing trivalent (+3) arsenic are much more toxic that those containing pentavalent (+5) arsenic. The valency of the arsenic atom is a more important factor in determining toxicity than the organic or inorganic nature of the arsenic-containing compound (Edmonds and Francesconi, 1993). With respect to inorganic arsenic compounds, salts of arsenic acid (arsenates) with arsenic in the pentavalent state are less toxic than arsenite compounds with arsenic in the trivalent state (Penrose, 1974). Because some reduction of arsenate (pentavalent arsenic) to arsenite (trivalent arsenic) might occur in the mammalian body (Vahter and Envall, 1983), it would be unwise to disregard the possible toxicity

of inorganic arsenic ingested in either valency state (Edmonds and Francesconi, 1993).

Seafood is a major source of trace amounts of arsenic in the human diet. However, arsenic in the edible parts of fish and shellfish is predominantly present as the arsenic-containing organic compound arsenobetaine (Cullen and Reimer, 1989; Edmonds and Francesconi, 1987a; NAS, 1991). Arsenobetaine is a stable compound containing a pentavalent arsenic atom, which has been shown to be metabolically inert and nontoxic in a number of studies (Cannon et al., 1983; Jongen et al., 1985; Kaise et al., 1985; Sabbioni et al., 1991; Vahter et al., 1983), and is not generally considered a threat to human health (ATSDR, 1989). Inorganic arsenic, although a minor component of the total arsenic content of fish and shellfish when compared to arsenobetaine, presents potential toxicity problems. To the degree that inorganic forms of arsenic are either present in seafood or, upon consumption, may be produced as metabolites of organic arsenic compounds in seafood, some human health risk, although small, would be expected (NAS, 1991).

Inorganic arsenic is very toxic to mammals and has been assigned to Toxicity Class I based on oral toxicity tests (*Farm Chemicals Handbook*, 1989). Use of several arsenical pesticides has been discontinued because of the health risks to animals and man. Inorganic arsenic also has been classified as a human carcinogen (A) (IRIS, 1995) and long-term effects include dermal hyperkeratosis, dermal melanosis and carcinoma, hepatomegaly, and peripheral neuropathy (NAS, 1991) (Appendix D).

Total arsenic (inclusive of both inorganic and organic forms) has been included in six national monitoring programs (Appendix C); however, no national program is currently monitoring total inorganic arsenic in fish or shellfish tissues. Arsenic and arsenic-containing organic compounds have not been shown to bioaccumulate to any great extent in aquatic organisms (NAS, 1977). Experimental evidence indicates that inorganic forms of both pentavalent and trivalent arsenic bioaccumulate minimally in several species of finfish including rainbow trout, bluegill, and fathead minnows (ASTER, 1995). A BCF value of 350 was reported for the American oyster (*Crassostrea virginica*) exposed to trivalent arsenic (Zaroogian and Hoffman, 1982). Only one State (Oregon) currently has an advisory in effect for arsenic contamination (RTI, 1993).

Edmonds and Francesconi (1993) summarized existing data from studies conducted outside the United States comparing concentrations of total arsenic, organic arsenic, and inorganic arsenic in marine fish and shellfish. Inorganic arsenic was found to represent from 0 to 44 percent of the total arsenic in marine fish and shellfish species surveyed. Residue concentrations of inorganic arsenic in the tissues typically ranged from 0 to 5.6 ppm (wet weight basis); but were generally less than 0.5 ppm for most species. In a study of six species of freshwater fish monitored as part of the Lower Columbia River study, inorganic arsenic represented from 0.1 to 27 percent of the total arsenic, and tissue residues of inorganic arsenic ranging from 0.001 to 0.047 ppm (wet weight) were 100 times lower than those reported for marine species (Tetra Tech, 1995).

Because it is the concentration of inorganic arsenic in fish and shellfish that poses the greatest threat to human health, EPA recommends that total inorganic arsenic

(not total arsenic) be analyzed in contaminant monitoring programs. Total inorganic arsenic should be considered for inclusion in State fish and shellfish monitoring programs in areas where its use is or has been extensive. States should contact their appropriate State agencies to obtain information on the historic and current uses of arsenic particularly as a wood preservative and in agricultural pesticides.

4.3.1.2 Cadmium—

Cadmium is commonly found in zinc, lead, and copper deposits (May and McKinney, 1981). It is released into the environment from several anthropogenic sources: smelting and refining of ores, electroplating, application of phosphate fertilizers, surface mine drainage (U.S. EPA, 1978), and waste disposal operations (municipal incineration and land application) (U.S. EPA, 1979a, 1987c). Cadmium is also used in the manufacture of paints, alloys, batteries, and plastics and has been used in the control of moles and plant diseases in lawns.

Cadmium is a cumulative human toxicant; it has been shown to cause renal dysfunction and a degenerative bone disease, itai-itai, in Japanese populations exposed via consumption of contaminated rice, fish, and water. Because cadmium is retained in the kidney, older individuals (over 40-50 years of age) typically have both the highest renal concentrations of cadmium and the highest prevalence of renal dysfunction (U.S. EPA, 1979a). Cadmium is a known carcinogen in animals, and there is limited evidence of the carcinogenicity of cadmium or cadmium compounds in humans. It has been classified as a probable human carcinogen by inhalation (B1) by EPA (IRIS, 1992).

Cadmium has been found to bioaccumulate in fish and shellfish tissues in fresh water (Schmitt and Brumbaugh, 1990) and in estuarine/marine waters (NOAA, 1987, 1989a) nationwide. In the National Contaminant Biomonitoring Program (NCBP), geometric mean concentrations of cadmium in freshwater fish were found to have declined from 0.07 ppm in 1976 to 0.03 ppm in 1984 (Schmitt and Brumbaugh, 1990). This trend contradicts the general trend of increasing cadmium concentrations in surface waters, which Smith et al. (1987) attribute to increasing U.S. coal combustion (Schmitt and Brumbaugh, 1990). Two States (New York and Ohio) have issued advisories for cadmium contamination (RTI, 1993).

Cadmium should be considered for inclusion in all State fish and shellfish contaminant monitoring programs.

4.3.1.3 Mercury—

The major source of atmospheric mercury is the natural degassing of the earth's crust, amounting to 2,700 to 6,000 tons per year (WHO, 1990). Primary points of entry of mercury into the environment from anthropogenic sources are industrial discharges and wastes (e.g., the chlorine-alkali industry) and atmospheric deposition resulting from combustion of coal and municipal refuse incinerators (Glass et al., 1990). Primary industrial uses of mercury are in the manufacture of batteries, vapor discharge lamps, rectifiers, fluorescent bulbs, switches, thermometers, and industrial control instruments (May and McKinney, 1981), and these products ultimately end up in landfills or incinerators. Mercury has also been used as a slimicide in the pulp and paper industry, as an antifouling and mildew-proofing agent in paints, and as an

antifungal seed dressing and in chlor-alkali production facilities (*Farm Chemicals Handbook*, 1989; Friberg and Vostal, 1972).

Although mercury use and losses from industrial processes in the United States have been reduced significantly since the 1970s, mercury contamination associated with increased fossil fuel combustion is of concern in some areas and may pose more widespread contamination problems in the future. An estimated 5,000 tons of mercury per year are released into the environment from fossil fuel burning (Klaassen et al., 1986). There is also increasing evidence of elevated mercury concentrations in areas where acid rain is believed to be a factor, although the extent of this problem has not been documented with certainty (Sheffy, 1987; Wiener, 1987). Volatilization from surfaces painted with mercury-containing paints, both indoors and outdoors, may have been a significant source in the past (Agocs et al., 1990; Sheffy, 1987). The United States estimated that 480,000 pounds of mercuric fungicides were used in paints and coatings in 1987 (NPCA, 1988). In July 1990, EPA announced an agreement with the National Paint and Coatings Association to cancel all registrations for use of mercury or mercury compounds in interior paints and coatings. In May 1991, the paint industry voluntarily canceled all remaining registrations for mercury in exterior paints.

Cycling of mercury in the environment is facilitated by the volatile character of its metallic form and by bacterial transformation of metallic and inorganic forms to stable alkyl mercury compounds, particularly in bottom sediments, which leads to bioaccumulation of mercury (Wood, 1974). Practically all mercury in fish tissue is in the form of methylmercury, which is toxic to humans (NAS, 1991; Tollefson, 1989).

The EPA has determined that the evidence of carcinogenicity of mercury in both animals and humans is inadequate and has assigned this metal a D carcinogenicity classification (IRIS, 1992). Both inorganic and organic forms of mercury are neurotoxicants. Fetuses exposed to organic mercury have been found to be born mentally retarded and with symptoms similar to those of cerebral palsy (Marsh, 1987). Individuals exposed to mercury via long-term ingestion of mercury-contaminated fish have been found to exhibit a wide range of symptoms, including numbness of the extremities, tremors, spasms, personality and behavior changes, difficulty in walking, deafness, blindness, and death (U.S. EPA, 1981a). Organomercury compounds were the causative agents of Minamata Disease, a neurological disorder reported in Japan during the 1950s among individuals consuming contaminated fish and shellfish (Kurland et al., 1960), with infants exposed prenatally found to be at significantly higher risk than adults. The EPA is especially concerned about evidence that the fetus is at increased risk of adverse neurological effects from exposure to methylmercury (e.g., Marsh et al., 1987; Piotrowski and Inskip, 1981; Skerfving, 1988; WHO, 1976, 1990).

Mercury has been found in both fish and shellfish from estuarine/marine (NOAA, 1987, 1989a) and fresh waters (Schmitt and Brumbaugh, 1990) at diverse locations nationwide. In contrast to cadmium and selenium, concentrations of mercury in freshwater fish tissue did not change between 1976 and 1984 (Schmitt and Brumbaugh, 1990). Mercury, the only metal analyzed in the National Study of Chemical Residues in Fish, was detected at 92.2 percent of 374 sites surveyed. Maximum, arithmetic mean, and median concentrations in fish tissue were 1.80, 0.26, and 0.17 ppm, respectively (U.S. EPA, 1991h, 1992c, 1992d). Fish

consumption advisories have been issued in 27 States as a result of mercury contamination (see Figure 4-1). In particular, mercury is responsible for a large number of the fish advisories currently in effect for lakes in Wisconsin, Michigan, and Minnesota and for rivers and lakes in Florida (RTI, 1993).

Mercury should be considered for inclusion in all State fish and shellfish contaminant monitoring programs. Only two national programs (301(h) and the FDA) currently analyze specifically for methylmercury; however, six programs analyze for total mercury (Appendix C). Because of the higher cost of methylmercury analysis, EPA recommends that total mercury be determined in State fish contaminant monitoring programs and the conservative assumption be made that all mercury is present as methylmercury in order to be most protective of human health.

It should be noted that Bache et al. (1971) analyzed methylmercury concentrations in lake trout of known ages and found that methylmercury concentration and the ratio of methylmercury to total mercury increased with age. Relative proportions of methylmercury in fish varied between 30 and 100 percent, with methylmercury concentrations lower than 80 percent occurring in fish 3 years of age or younger. Thus, when high concentrations of total mercury are detected, and if resources are sufficient, States may wish to repeat sampling and obtain more specific information on actual concentrations of methylmercury in various age or size classes of fish.

4.3.1.4 Selenium—

Selenium is a natural component of many soils, particularly in the west and southwest regions of the United States (NAS, 1991). It enters the environment primarily via emissions from oil and coal combustion (May and McKinney, 1981; Pillay et al., 1969). Selenium is an essential nutrient but is toxic to both humans and animals at high concentrations and has been shown to act as a mutagen in animals (NAS, 1991). Long-term adverse effects from ingestion by humans have not been studied thoroughly. The EPA has determined that the evidence of carcinogenicity of selenium in both humans and animals is inadequate and, therefore, has assigned this metal a D carcinogenicity classification. However, selenium sulfide has been classified as a probable human carcinogen (B2) (IRIS, 1992).

Selenium is frequently detected in ground and surface waters in most regions of the United States and has been detected in marine fish and shellfish (NOAA, 1987, 1989a) and in freshwater fish (Schmitt and Brumbaugh, 1990) from several areas nationwide. Selenium has been monitored in five national fish contaminant monitoring programs (Appendix C). Definitive information concerning the chemical forms of selenium found in fish and shellfish is not available (NAS, 1976, 1991). Five States (California, Colorado, North Carolina, Texas, and Utah) have issued advisories for selenium contamination in fish (RTI, 1993).

Selenium should be considered for inclusion in all State fish and shellfish monitoring programs.

4.3.1.5 Tributyltin

Tributyltin belongs to the organometallic family of tin compounds that have been used as biocides, disinfectants, and antifoulants. Antifoulant paints containing

tributyltin compounds were first registered for use in the United States in the early 1960s. Tributyltin compounds are used in paints applied to boat and ship hulls as well as to crab pots, fishing nets, and buoys to retard the growth of fouling organisms. These compounds are also registered for use as wood preservatives, disinfectants, and biocides in cooling towers, pulp and paper mills, breweries, leather processing facilities, and textile mills (U.S. EPA, 1988c).

Tributyltin compounds are acutely toxic to aquatic organisms at concentrations below 1 ppb and are chronically toxic to aquatic organisms at concentrations as low as 0.002 ppb (U.S. EPA, 1988c). The Agency initiated a Special Review of tributyltin compounds used as antifoulants in January of 1986 based on concerns over its adverse effects on nontarget aquatic species. Shortly thereafter the Organotin Antifouling Paint Control Act (OAPCA) was enacted in June 1988, which contained interim and permanent tributyltin use restrictions as well as environmental monitoring, research, and reporting requirements. The Act established interim release rate restrictions under which only tributyltin-containing products that do not exceed an average daily release rate of 4 µg organotin/cm^2/day can be sold or used. The OAPCA also contained a permanent provision to prohibit the application of tributyltin antifouling paints to non-aluminum vessels under 25 meters (82 feet) long (U.S. EPA, 1988c).

Tributyltin compounds are highly toxic to mammals (i.e., LD_{50} values ranged from 0.04 to 60 mg/kg) based on animal testing (Eisler, 1989; IRIS, 1995). Immunotoxicity and neurotoxicity are the critical effects produced by tributyltin. Insufficient data are available to evaluate the carcinogenicity of tributyltin compounds (IRIS, 1995) (Appendix D).

Tributyltins have been found to bioaccumulate in fish, bivalve mollusks, and crustaceans. Bioconcentration factors (BCF) ranging from 200 to 4,300 for finfish, from 2,000 to 6,000 for bivalves, and of 4,400 have been reported for crustaceans (U.S. EPA, 1988c). Tributyltin used to control marine fouling organisms in an aquaculture rearing pen has been found to bioaccumulate in fish tissue (Short and Thrower, 1987a and 1987b). Tsuda et al. (1988) reported a BCF value of 501 for tributyltin in carp (*Cyprinus carpio*) muscle tissue. Martin et al. (1989) reported a similar BCF value of 406 for tributyltin in rainbow trout (*Salmo gairdneri*) and Ward et al. (1981) reported a BCF value of 520 for the sheepshead minnow (*Cyprinodon variegatus*). In an environmental monitoring study conducted in England, a BCF value of 1,000 was reported for tributyltin in seed oysters (*Crassostrea gigas*) (Ebdon et al., 1989).

Tributyltin is recommended for monitoring by the FDA but has not been monitored in any other national fish contaminant monitoring program (Appendix C). Only one State, Oregon, currently has an advisory in effect for tributyltin contamination in shellfish (RTI, 1993).

Tributyltin should be considered for inclusion in all State fish and shellfish contaminant monitoring programs, particularly in States with coastal waters, States bordering the Great Lakes, or States with large rivers where large ocean- going vessels are used for commerce. Tributyltin concentrations are reported to be highest in areas of heavy boating and shipping activities including shipyards where

tributyltin-containing antifouling paints are often removed and reapplied. Before recoating, old paint containing tributyltin residues is scraped from the vessel hull and these paint scrapings are sometimes washed into the water adjacent to the boat or shipyard despite the tributyltin label prohibiting this practice (U.S. EPA, 1988c). Tributyltin should be considered for inclusion in State fish and shellfish monitoring programs in areas where its use is or has been extensive. States should contact their appropriate agencies to obtain information on the historic and current uses of tributyltin, particularly with respect to its uses in antifouling paints and wood preservatives.

4.3.2 Organochlorine Pesticides

The following organochlorine pesticides and metabolites are recommended as target analytes in screening studies: total chlordane (sum of cis- and trans-chlordane, cis- and trans-nonachlor, and oxychlordane), total DDT (sum of 2,4'- and 4,4'-isomers of DDT, DDD, and DDE), dicofol, dieldrin, endosulfan I and II, endrin, heptachlor epoxide, hexachlorobenzene, lindane (γ-hexachlorocyclohexane), mirex, and toxaphene (see Appendix D). Mirex is of particular concern in the Great Lakes States and the southeast States (NAS, 1991). All of these compounds are neurotoxins and most are known or suspected human carcinogens (IRIS, 1992; Sax, 1984).

With the exception of endosulfan I and II, dicofol and total DDT, each of the pesticides on the recommended target analyte list (Table 4-1) has been included in at least five major fish contaminant monitoring programs (Appendix C), and seven of the compounds have triggered at least one State fish consumption advisory (Table 4-2). Although use of some of these pesticides has been terminated or suspended within the United States for as long as 20 years (Appendix D), these compounds still require long-term monitoring. Many of the organochlorine pesticides were used in large quantities for over a decade and are present in sediments at high concentrations. Organochlorine pesticides are not easily degraded or metabolized and, therefore, persist in the environment. These compounds are either insoluble or have relatively low solubility in water but are quite lipid soluble. Because these compounds are not readily metabolized or excreted from the body and are readily stored in fatty tissues, they can bioaccumulate to high concentrations through aquatic food chains to secondary consumers (e.g., fish, piscivorous birds, and mammals including humans).

Pesticides may enter aquatic ecosystems from point source industrial discharges or from nonpoint sources such as aerial drift and/or runoff from agricultural use areas, leaching from landfills, or accidental spills or releases. Agricultural runoff from crop and grazing lands is considered to be the major source of pesticides in water, with industrial waste (effluents) from pesticide manufacturing the next most common source (Li, 1975). Significant atmospheric transport of pesticides to aquatic ecosystems can also result from aerial drift of pesticides, volatilization from applications in terrestrial environments, and wind erosion of treated soil (Li, 1975). Once in water, pesticide residues may become adsorbed to suspended material, deposited in bottom sediment, or absorbed by organisms in which they are detoxified and eliminated or accumulated (Nimmo, 1985).

The reader should note that two of the organochlorine pesticides have active registrations: endosulfan and dicofol. States should contact their appropriate State agencies to obtain information on both the historic and current uses of these pesticides.

4.3.2.1 Chlordane (Total)—

Chlordane is a multipurpose insecticide that has been used extensively in home and agricultural applications in the United States for the control of termites and many other insects (Appendix D). This pesticide is similar in chemical structure to dieldrin, although less toxic (Toxicity Class II), and has been classified as a probable human carcinogen (B2) by EPA (IRIS, 1992; Worthing, 1991).

Although the last labeled use of chlordane as a termiticide was phased out in the United States beginning in 1975, it has been monitored in eight national fish contaminant programs (Appendix C) and has been widely detected in freshwater fish (Schmitt et al., 1990) and in both estuarine/marine finfish (NOAA, 1987) and marine bivalves (NOAA, 1989a) at concentrations of human health concern. The cis- and trans-isomers of chlordane and nonachlor, which are primary constituents of technical-grade chlordane, and oxychlordane, the major metabolite of chlordane, were monitored as part of the National Study of Chemical Residues in Fish. These compounds were detected in fish tissue at the following percentage of the 362 sites surveyed: cis-chlordane (64 percent), trans-chlordane (61 percent), cis-nonachlor (35 percent), trans-nonachlor (77 percent), and oxychlordane (27 percent) (U.S. EPA, 1992c, 1992d). Chlordane's presence in fish tissue has resulted in consumption advisories in 24 States (see Figure 4-2).

Total chlordane (i.e., sum of cis- and trans-chlordane, cis- and trans-nonachlor, and oxychlordane) should be considered for inclusion in all State fish and shellfish contaminant monitoring programs (NAS, 1991).

4.3.2.2 DDT (Total)—

Although the use of DDT was terminated in the United States in 1972, DDT and its DDE and DDD metabolites persist in the environment and are known to bioaccumulate (Ware, 1978). DDT, DDD, and DDE have all been classified by EPA as probable human carcinogens (B2) (IRIS, 1992).

DDT or its metabolites have been included as target analytes in eight major fish and shellfish monitoring programs (Appendix C) and contamination has been found to be widespread (NOAA, 1987, 1989a; Schmitt et al., 1990). DDE, the only DDT metabolite surveyed in fish tissue in the National Study of Chemical Residues in Fish, was detected at more sites than any other single pollutant (99 percent of the 362 sites sampled) (U.S. EPA, 1992c, 1992d). Nine States (Alabama, American Samoa, Arizona, California, Delaware, Massachusetts, Nebraska, New York, and Texas) currently have fish consumption advisories in effect for DDT or its metabolites (RTI, 1993).

Total DDT (i.e., sum of the 4,4'- and 2,4'- isomers of DDT and of its metabolites, DDE and DDD) should be considered for inclusion in all State fish and shellfish contaminant monitoring programs.

4.3.2.3 Dicofol—

This chlorinated hydrocarbon acaricide was first registered in 1957 and is structurally similar to DDT (U.S. EPA, 1992c, 1992d). Technical-grade dicofol may contain impurities of the p,p' and o,p' isomers of DDT, DDE, DDD, and a compound known as extra-chlorine DDT (Cl-DDT) that are inherent as a result of the manufacturing process (U.S. EPA, 1983b). Historically, dicofol has been used to control mites on cotton and citrus (60 percent), on apples (10 percent), on ornamental plants and turf (10 percent), and on a variety of other agricultural products (20 percent) including pears, apricots, and cherries (*Farm Chemical Handbook*, 1989), as a seed crop soil treatment, on vegetables (e.g., beans and corn) and on shade trees (U.S. EPA, 1992c, 1992d).

Dicofol is moderately toxic to laboratory rats and has been assigned to Toxicity Class III based on oral exposure studies (Appendix D). Technical-grade dicofol induced hepatocellular (liver) carcinomas in male mice; however, results were negative in female mice and in rats (NCI, 1978). EPA has classified dicofol as a possible human carcinogen (C) (U.S. EPA, 1992a). Because of concern that dicofol would have the same effect as DDT on thinning of egg shells, the FDA required all dicofol products to contain less than 0.1 percent DDT and related contaminants after June 1, 1989 (51 FR 19508).

Dicofol was recommended for monitoring by the EPA Office of Water as part of the Assessment and Control of Bioconcentratable Contaminants in Surface Waters Program and has been included in two national monitoring programs (see Appendix C). In the National Study of Chemical Residues in Fish, dicofol was detected at 16 percent of the sites monitored (U.S. EPA, 1992c, 1992d). Dicofol concentrations were greater than the quantification limit (2.5 ppb) in samples from 7 percent of the sites. Most of the sites where dicofol was detected were in agricultural areas where citrus and other fruits and vegetables are grown (U.S. EPA, 1992c, 1992d). It should be noted that this national study did not specifically target agricultural sites where this pesticide historically had been or currently is used. Dicofol residues in fish could be much higher if sampling were targeted for pesticide runoff. Experimental evidence indicates this compound bioaccumulates in Bluegill sunfish (BCF from 6,600 to 17,000) (U.S. EPA, 1993a); however, no consumption advisories are currently in effect for dicofol (RTI, 1993).

Dicofol should be considered for inclusion in State fish and shellfish contaminant monitoring programs, in areas where its use is or has been extensive. States should contact their appropriate State agencies to obtain information on the historic and current uses of this pesticide.

4.3.2.4 Dieldrin—

Dieldrin is a chlorinated cyclodiene that was widely used in the United States from 1950 to 1974 as a broad spectrum pesticide, primarily on termites and other soil-dwelling insects and on cotton, corn, and citrus crops. Because the toxicity of this persistent pesticide posed an imminent danger to human health, EPA banned the production and most major uses of dieldrin in 1974, and, in 1987, all uses of dieldrin were voluntarily canceled by industry (see Appendix D).

Dieldrin has been classified by EPA as a probable human carcinogen (B2) (IRIS, 1992) and has been identified as a human neurotoxin (ATSDR, 1987a). Dieldrin has been included in eight national minitoring programs (Appendix C) and is still detected nationwide in freshwater finfish (Schmitt et al., 1990) and estuarine/marine finfish and shellfish (NOAA, 1987, 1989a). Dieldrin was detected in fish tissue at 60 percent of the 362 sites surveyed as part of the National Survey of Chemical Residues in Fish (U.S. EPA, 1992c, 1992d). Because it is a metabolite of aldrin, the environmental concentrations of dieldrin are a cumulative result of the historic use of both aldrin and dieldrin (Schmitt et al., 1990). Three States (Arizona, Illinois, and Nebraska) have issued advisories for dieldrin contamination in fish (RTI, 1993).

Dieldrin should be considered for inclusion in all State fish and shellfish contaminant monitoring programs.

4.3.2.5 Endosulfan—

Endosulfan is a chlorinated cyclodiene pesticide that is currently in wide use primarily as a noncontact insecticide for seed and soil treatments (Appendix D). Two stereoisomers (I and II) exist and exhibit approximately equal effectiveness and toxicity (Worthing, 1991).

Endosulfan is highly toxic to humans and has been assigned to Toxicity Class I. To date, no studies have been found concerning carcinogenicity in humans after oral exposure to endosulfan (ATSDR, 1990). EPA has given endosulfan the carcinogenicity classification E, indicating there is no evidence of carcinogenicity for humans (U.S. EPA, 1992a).

Agricultural runoff is the primary source of this pesticide in aquatic ecosystems. Endosulfan has been shown to be highly toxic to fish and marine invertebrates and is readily absorbed in sediments. It therefore represents a potential hazard in the aquatic environment (Sittig, 1980). However, data are currently insufficient to assess nationwide endosulfan contamination (NAS, 1991). Endosulfan was recommended for monitoring by the FDA and has been included in one national fish contaminant monitoring program (U.S. EPA 301(h) Program) evaluated by the EPA Workgroup (Appendix C). No consumption advisories are currently in effect for endosulfan I or II (RTI, 1993).

Endosulfan I and II should be considered for inclusion in all State fish and shellfish contaminant monitoring programs in areas where its use is or has been extensive. States should contact their appropriate State agencies to obtain information on the historic and current uses of this pesticide.

4.3.2.6 Endrin—

Endrin is a chlorinated cyclodiene that historically was widely used as a broad spectrum pesticide. Endrin was first registered for use in the United States in 1951. However, recognition of its long-term persistence in soil and its high levels of mammalian toxicity led to restriction of its use beginning in 1964 (U.S. EPA, 1980a) and 1979 (44 FR 43632) and to final cancellation of its registration in 1984 (U.S. EPA, 1984a) (Appendix D).

Endrin is highly toxic to humans (Toxicity Class I), with acute exposures affecting the central nervous system primarily (Sax, 1984). At present, evidence of both animal and human carcinogenicity of endrin is considered inadequate (IRIS, 1992).

Although endrin has been included in six national fish contaminant monitoring programs (Appendix C), it has not been found widely throughout the United States. Endrin was detected in freshwater and marine species at 11 percent of the 362 sites surveyed in the EPA National Study of Chemical Residues in Fish (U.S. EPA, 1992c, 1992d) and was found in only 29 percent of 112 stations sampled in the NCBP (Schmitt et al., 1990). No States have issued fish consumption advisories for endrin (RTI, 1993).

Endrin should be considered for inclusion in all State fish and shellfish contaminant monitoring programs.

4.3.2.7 Heptachlor Epoxide—

Heptachlor epoxide is not a formulated pesticide, but is a metabolic degradation product of the pesticide heptachlor. It is also found as a contaminant in heptachlor and chlordane formulations (Appendix D). Heptachlor has been used as a persistent, nonsystemic contact and ingested insecticide on soils (particularly for termite control) and seeds and as a household insecticide (Worthing, 1991). EPA suspended the major uses of heptachlor in 1978 (ATSDR, 1987b). Acute exposures to high doses of heptachlor epoxide in humans can cause central nervous system effects (e.g., irritability, dizziness, muscle tremors, and convulsions (U.S. EPA, 1986e). In animals, liver, kidney, and blood disorders can occur (IRIS, 1989). Exposure to this compound produced an increased incidence of liver carcinomas in rats and mice and hepatomas in female rats (IRIS, 1989). Heptachlor epoxide has been classified by EPA as a probable human carcinogen (B2) (IRIS, 1992).

Heptachlor epoxide has been included in seven national fish monitoring programs (Appendix C) and has been detected widely in freshwater finfish (Schmitt et al., 1990) but infrequently in bivalves and marine fish (NOAA, 1987, 1989a). Heptachlor epoxide was detected in fish tissue at 16 percent of the 362 sites where it was surveyed in the National Study of Chemical Residues in Fish (U.S. EPA, 1992c, 1992d). One State (Nebraska) currently has fish advisories for heptachlor epoxide contamination (RTI, 1993).

Heptachlor epoxide should be considered for inclusion in all State fish and shellfish monitoring programs.

4.3.2.8 Hexachlorobenzene—

Hexachlorobenzene is a fungicide that was widely used as a seed protectant in the United States until 1985 (Appendix D). The use of hexachlorobenzene and the presence of hexachlorobenzene residues in food are banned in many countries including the United States (Worthing, 1991). Registration of hexachlorobenzene as a pesticide was voluntarily canceled in 1984 (Morris and Cabral, 1986).

The toxicity of this compound is minimal; it has been given a toxicity classification of IV (i.e., oral LD_{50} greater than 5,000 ppm in laboratory animals (*Farm Chemicals*

Handbook, 1989). However, nursing infants are particularly susceptible to hexachlorobenzene poisoning as lactational transfer can increase infant tissue levels to levels two to five times maternal tissue levels (ATSDR, 1989b). Hexachlorobenzene is a known animal carcinogen (ATSDR, 1989b) and has been classified by EPA as a probable human carcinogen (B2) (IRIS, 1992) (Appendix D).

Of the chlorinated benzenes, hexachlorobenzene is the most widely monitored (Worthing, 1991). It was included as a target analyte in seven of the major monitoring programs reviewed (Appendix C). Hexachlorobenzene was detected in fish tissue at 46 percent of the 362 sites where it was surveyed in the National Study of Chemical Residues in Fish (U.S. EPA, 1992c, 1992d). Two States (Louisiana and Ohio) have issued advisories for hexachlorobenzene contamination in fish and shellfish (RTI, 1993).

Hexachlorobenzene should be considered for inclusion in all State fish and shellfish monitoring programs.

4.3.2.9 Lindane—

Lindane is a mixture of isomers of hexachlorocyclohexane ($C_6H_6Cl_6$), whose major component ($\geq$99 percent) is the gamma isomer. It is commonly referred to as either γ-HCH (hexachlorocyclohexane) or γ-BHC (benzene hexachloride). Lindane is used primarily in seed treatments, soil treatments for tobacco transplants, foliage applications on fruit and nut trees and vegetables, and wood and timber protection. Since 1985, many uses of lindane have been banned or restricted (see Appendix D). At present, its application is permitted only under supervision of a certified applicator (U.S. EPA, 1985c).

Lindane is a neurotoxin (assigned to Toxicity Class II) and has been found to cause aplastic anemia in humans (Worthing, 1991). Lindane has been classified by EPA as a probable/possible human carcinogen (B2/C). Available data for this pesticide need to be reviewed, but at a minimum the carcinogenicity classification will be C (U.S. EPA, 1992a).

Lindane has been included in eight major fish contaminant monitoring programs (Appendix C). This pesticide has been detected in freshwater fish (Schmitt et al., 1990) and in marine fish and bivalves (NOAA, 1987, 1989a) nationwide. Lindane was detected in fish tissue at 42 percent of 362 sites surveyed in the National Study of Chemical Residues in Fish (U.S. EPA, 1992c, 1992d). Although lindane has been widely monitored and widely detected, no consumption advisories are currently in effect for lindane (RTI, 1993).

Lindane should be considered for inclusion in all State fish and shellfish monitoring programs.

4.3.2.10 Mirex—

Mirex is a chlorinated cyclodiene pesticide that was used in large quantities in the United States from 1962 through 1975 primarily for control of fire ants in the Southeast and, more widely, under the name Dechlorane as a fire retardant and polymerizing agent in plastics (Kaiser, 1978) (Appendix D).

Mirex has been assigned to Toxicity Class II and has been classified as a probable human carcinogen by the International Agency for Research on Cancer (IARC, 1987); however, the carcinogenicity data are currently under review by EPA (IRIS, 1992). EPA instituted restrictions on the use of mirex in 1975, and, shortly thereafter, the U.S. Department of Agriculture (USDA) suspended the fire ant control program (Hodges, 1977).

Mirex has been included in eight major fish contaminant monitoring programs (Appendix C). It has been found primarily in the Southeast and the Great Lakes regions (NAS, 1991; Schmitt et al., 1990). Mirex was detected in fish tissue at 36 percent of 362 sites surveyed in the National Study of Chemical Residues in Fish (U.S. EPA, 1992c, 1992d). Three States (New York, Ohio, and Pennsylvania) currently have fish consumption advisories for mirex (RTI, 1993).

Mirex should be considered for inclusion in all State fish and shellfish monitoring programs.

4.3.2.11 Toxaphene—

Toxaphene is a mixture of chlorinated camphenes. Historically, it was used in the United States as an insecticide primarily on cotton (Hodges, 1977). Partly as a consequence of the ban on the use of DDT imposed in 1972, toxaphene was for many years the most heavily used pesticide in the United States (Eichers et al., 1978). In 1982, toxaphene's registration for most uses was canceled (47 FR 53784).

Like many of the other organochlorine pesticides, toxaphene has been assigned to Toxicity Class II (Appendix D). Unlike the other organochlorine pesticides, toxaphene is fairly easily metabolized by mammals and is not stored in the fatty tissue to any great extent. Toxaphene has been classified by EPA as a probable human carcinogen (B2) (IRIS, 1992).

Toxaphene has been included in five major fish contaminant monitoring programs (Appendix C). It has been detected frequently in both fresh (Schmitt et al., 1990) and estuarine (NOAA, 1989a) waters but is only consistently found in Georgia, Texas, and California (NAS, 1991). **Note:** A toxaphene-like compound that is a byproduct of the paper industry has been identified in the Great Lakes Region (J. Hesse, Michigan Department of Public Health, personal communication, 1993). Two States (Arizona and Texas) currently have fish advisories in effect for toxaphene (RTI, 1993).

Toxaphene should be considered for inclusion in all State fish and shellfish monitoring programs.

4.3.3 Organophosphate Pesticides

The following organophosphate pesticides are recommended as target analytes in screening studies: chlorpyrifos, diazinon, disulfoton, ethion, and terbufos (Appendix D). Most of these organophosphate pesticides share two distinct features. Organophosphate pesticides are generally more toxic to vertebrates than organochlorine pesticides and exert their toxic action by inhibiting the activity of

cholinesterase (ChE), one of the vital nervous system enzymes. In addition, organophosphates are chemically unstable and thus are less persistent in the environment. It is this latter feature that made them attractive alternatives to the organochlorine pesticides that were used extensively in agriculture from the 1940s to the early 1970s.

With the exception of chlorpyrifos, none of the organophosphates has been included in any of the national fish contaminant monitoring programs evaluated by the EPA Workgroup and none of these pesticides (including chlorpyrifos) has triggered State fish consumption advisories. All of the compounds have active pesticide registrations and have been recommended for monitoring because they have a Toxicity Classification of I or II (Appendix D), have BCFs > 300, a half-life of 30 days or more in the environment, and their use profiles suggest they could be potential problems in some agricultural watersheds.

The reader should note that all of the organophosphate pesticides recommended as target analytes have active registrations. States should contact their appropriate State agencies to obtain information on both the historic and current uses of these pesticides. In addition, if a State determines that use of these pesticides may be occurring in its waters, sampling should be conducted during late spring or early summer within 1 to 2 months following pesticide application because these compounds are degraded and metabolized relatively rapidly by fish species. Additional discussion of appropriate sampling times for fish contaminant monitoring programs is provided in Section 6.1.1.5.

4.3.3.1 Chlorpyrifos—

This organophosphate pesticide was first introduced in 1965 to replace the more persistent organochlorine pesticides (e.g., DDT) (U.S. EPA, 1986e) and has been used for a broad range of insecticide applications (Appendix D). Chlorpyrifos is used primarily to control soil and foliar insects on cotton, peanuts, and sorghum (Worthing, 1991; U.S. EPA, 1986e). Chlorpyrifos is also used to control root-infesting and boring insects on a variety of fruits (e.g., apples, bananas, citrus, grapes), nuts (e.g., almonds, walnuts), vegetables (e.g., beans, broccoli, brussel sprouts, cabbage, cauliflower, peas, and soybeans), and field crops (e.g., alfalfa and corn) (U.S. EPA, 1984c). As a household insecticide, chlorpyrifos has been used to control ants, cockroaches, fleas, and mosquitoes (Worthing, 1991) and is registered for use in controlling subsurface termites in California (U.S. EPA, 1983a). Based on use application, 57 percent of all chlorpyrifos manufactured in the United States is used on corn, while 22 percent is used for pest control and lawn and garden services (U.S. EPA, 1993a).

Chlorpyrifos has a moderate mammalian toxicity and has been assigned to Toxicity Class II based on oral feeding studies (*Farm Chemicals Handbook*, 1989). No teratogenic or fetotoxic effects were found in mice or rats (IRIS, 1989). No carcinogenicity was found in chronic feeding studies with rats, mice and dogs (U.S. EPA, 1983a). EPA has assigned chlorpyrifos a carcinogenicity classification of D—not classifiable based on inadequate evidence of carcinogenicity or lack of data in at least two animal studies or in both epidemiologic and animal studies (U.S. EPA, 1992a).

Chlorpyrifos was recommended for monitoring by the FDA and has been included in one national monitoring program, the National Study of Chemical Residues in Fish (see Appendix C). In this latter study, chlorpyrifos was detected at 26 percent of sites sampled nationally (U.S. EPA, 1992c, 1992d). Eighteen percent of the sites with relatively high concentrations (2.5 to 344 ppb) were scattered throughout the East, Midwest, and in California; the highest concentrations detected (60 to 344 ppb) were found either in agricultural areas or in urban areas with a variety of nearby industrial sources. It should be noted that this national study did not specifically target agricultural sites where this pesticide historically had been used or is currently used. Chlorpyrifos residues in fish could be much higher if sampling were targeted for pesticide runoff. Experimental evidence indicates this compound bioaccumulates in rainbow trout (BCF from 1,280 to 3,903) (U.S. EPA, 1993a); however, no consumption advisories are currently in effect for chlorpyrifos (RTI, 1993).

Chlorpyrifos should be considered for inclusion in State fish and shellfish contaminant monitoring programs in areas where its use is or has been extensive. States should contact their appropriate State agencies to obtain information on the historic and current uses of this pesticide.

4.3.3.2 Diazinon—

Diazinon is a phosphorothiate insecticide and nematicide that was first registered in 1952 for control of soil insects and pests of fruits, vegetables, tobacco, forage, field crops, range, pasture, grasslands, and ornamentals; for control of cockroaches and other household insects; for control of grubs and nematodes in turf; as a seed treatment; and for fly control (U.S. EPA, 1986f).

Diazinon is moderately toxic to mammals and has been assigned to Toxicity Class II based on oral toxicity tests (Appendix D). EPA has assigned diazinon to carcinogenicity classification D—not classifiable based on a lack of data or inadequate evidence of carcinogenicity in at least two animal tests or in both epidemiologic and animal studies (U.S. EPA, 1992a). This compound is also highly toxic to birds, fish, and other aquatic invertebrates (U.S. EPA, 1986f).

Diazinon has not been included in any national fish contaminant monitoring program evaluated by the EPA Workgroup (Appendix C). Experimental evidence indicates this compound accumulates in trout (BCF of 542) (U.S. EPA, 1993a); however, no consumption advisories are currently in effect for diazinon (RTI, 1993).

Diazinon should be considered for inclusion in State fish and shellfish contaminant monitoring programs in areas where its use is or has been extensive. States should contact their appropriate State agencies to obtain information on the historic and current uses of this pesticide.

4.3.3.3 Disulfoton—

Disulfoton is a multipurpose systemic insecticide and acaricide first registered in 1958 for use as a side dressing, broadcast, or foliar spray in the seed furrow to control many insect and mite species and as a seed treatment for sucking insects (Appendix D).

Disulfoton is highly toxic to all mammalian systems and has been assigned to Toxicity Class I on the basis of all routes of exposure (*Farm Chemicals Handbook*, 1989). All labeling precautions and use restrictions are based on human health risk. Disulfoton and its major metabolites are potent cholinesterase inhibitors primarily attacking acetylcholinesterase. Contradictory evidence is available on the mutagenicity of this compound and the EPA has concluded that the mutagenic potential is not adequately defined (U.S. EPA, 1984d). EPA has assigned disulfoton to carcinogenicity classification D—not classifiable based on a lack of data or inadequate evidence of carcinogenicity in at least two animal tests or in both epidemiologic and animal studies (U.S. EPA, 1992a).

Disulfoton has not been included in any national fish contaminant monitoring program evaluated by the EPA Workgroup (Appendix C). Experimental evidence indicates this compound accumulates in fish (BCF from 460 to 700) (U.S. EPA, 1993a); however, no consumption advisories are currently in effect for disulfoton (RTI, 1993).

Disulfoton should be considered for inclusion in State fish and shellfish contaminant monitoring programs in areas where its use is or has been extensive. States should contact their appropriate State agencies to obtain information on the historic and current uses of this pesticide.

4.3.3.4 Ethion—

Ethion is a multipurpose insecticide and acaricide that has been registered since 1965 for use on a wide variety of nonfood crops (turf, evergreen plantings, and ornamentals), food crops (seed, fruit, nut, fiber, grain, forage, and vegetables), and for domestic outdoor uses around dwellings and for lawns (Appendix D). Application to citrus crops accounts for 86 to 89 percent of the ethion used in the United States. The remaining 11 to 14 percent is applied to cotton and a variety of fruit and nut trees and vegetables. Approximately 55 to 70 percent of all domestically produced citrus fruits are treated with ethion (U.S. EPA, 1989e).

Acute oral toxicity studies have shown that technical-grade ethion is moderately toxic to mammals (Toxicity Class II) (*Farm Chemicals Handbook*, 1989). In a chronic rat toxicity study, a decrease in serum cholinesterase was observed in both males and females. Ethion was not found to be carcinogenic in rats and mice (U.S. EPA, 1989e). EPA has assigned ethion to carcinogenicity classification D—not classifiable based on a lack of data or inadequate evidence of carcinogenicity in at least two animal tests or in both epidemiologic and animal studies (U.S. EPA, 1992a).

Ethion has not been included in any national fish contaminant monitoring program evaluated by the EPA Workgroup (Appendix C). Experimental evidence indicates this compound accumulates in Bluegill sunfish (BCF from 880 to 2,400) (U.S. EPA, 1993a); however, no consumption advisories are currently in effect for ethion (RTI, 1993).

Ethion should be considered for inclusion in State fish and shellfish contaminant monitoring programs in areas where its use is or has been extensive. States should

contact their appropriate State agencies to obtain information on the historic and current uses of this pesticide.

4.3.3.5 Terbufos—

Terbufos is a systemic organophosphate insecticide and nematicide registered in 1974 principally for use on corn, sugar beets, and grain sorghum. The primary method of application involves direct soil incorporation of a granular formulation (*Farm Chemicals Handbook*, 1989).

Terbufos is highly toxic to humans and has been assigned to Toxicity Class I (Appendix D). Symptoms of acute cholinesterase inhibition have been reported in all acute studies, and cholinesterase inhibition was reported in several chronic mammalian feeding studies (U.S. EPA, 1985d). EPA has assigned terbufos to carcinogenicity classification D—not classifiable based on a lack of data or inadequate evidence of carcinogenicity in at least two animal tests or in both epidemiologic and animal studies (U.S. EPA, 1992a). Terbufos is also highly toxic to birds, fish, and other aquatic invertebrates (U.S. EPA, 1985d).

Terbufos has not been included in any national fish contaminant monitoring program evaluated by the EPA Workgroup (Appendix C). Experimental evidence indicates this compound accumulates in fish (BCF from 320 to 1,400) (U.S. EPA, 1993a); however no consumption advisories are currently in effect for terbufos (RTI, 1993).

Terbufos should be considered for inclusion in State fish and shellfish contaminant monitoring programs in areas where its use is or has been extensive. States should contact their appropriate State agencies to obtain information on the historic and current uses of this pesticide.

4.3.4 Chlorophenoxy Herbicides

Chlorophenoxy herbicides, which include oxyfluorfen, are nonselective foliar herbicides that are most effective in hot weather (Ware, 1978).

4.3.4.1 Oxyfluorfen—

Oxyfluorfen is a pre- and postemergence herbicide that has been registered since 1979 for use to control a wide spectrum of annual broadleaf weeds and grasses in apples, artichokes, corn, cotton, jojoba, tree fruits, grapes, nuts, soybeans, spearmint, peppermint, and certain tropical plantation and ornamental crops (Appendix D).

Evidence suggests that oxyfluorfen is moderately toxic to mammals and has been assigned to Toxicity Class II based on a chronic mouse feeding study (*Farm Chemicals Handbook*, 1989; IRIS, 1993). There is also evidence of carcinogenicity (liver tumors) in mice (U.S. EPA, 1993a) and therefore oxyfluorfen has been classified by EPA as a possible human carcinogen (C) (U.S. EPA, 1992c).

Although oxyfluorfen has an active registration, it has not been included in any national fish contaminant monitoring program evaluated by the EPA Workgroup

(Appendix C). Experimental evidence indicates this herbicide accumulates in Bluegill sunfish (BCF from 640 to 1,800) (U.S. EPA, 1993a); however, no consumption advisories are currently in effect for oxyfluorfen (RTI, 1993).

Oxyfluorfen should be considered for inclusion in State fish and shellfish contaminant monitoring programs in areas where its use is or has been extensive. States should contact their appropriate State agencies to obtain information on the historic and current uses of this pesticide.

4.3.5 Polycyclic Aromatic Hydrocarbons (PAHs)—

Polycyclic aromatic hydrocarbons are base/neutral organic compounds that have a fused ring structure of two or more benzene rings. PAHs are also commonly referred to as polynuclear aromatic hydrocarbons (PNAs). PAHs with two to five benzene rings (i.e., 10 to 24 skeletal carbons) are generally of greatest concern for environmental and human health effects (Benkert, 1992). These PAHs include those listed as priority pollutants (U.S. EPA, 1995a)

- Acenaphthene
- Acenaphthylene
- Anthracene
- Benz[*a*]anthracene
- Benzo[*a*]pyrene
- Benzo[*b*]fluoranthene
- Benzo[*k*]fluoranthene
- Benzo[*g,h,i*]perylene
- Chlorinated naphthalenes
- Chrysene
- Dibenz[*a,h*]anthracene
- Fluoranthene
- Fluorene
- Indeno[*1,2,3-cd*]pyrene
- Naphthalene
- Phenanthrene
- Pyrene.

The metabolites of many of the high-molecular-weight PAHs (e.g., benz[*a*]anthracene, benzo[*a*]pyrene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, chrysene, dibenz[*a,h*]anthracene, indeno[*1,2,3-cd*]pyrene) have been shown in laboratory test systems to be carcinogens, cocarcinogens, teratogens, and/or mutagens (Moore and Ramamoorthy, 1984; U.S. DHHS, 1990). Benzo[*a*]pyrene, one of the most widely occurring and potent PAHs, and several other PAHs (e.g., benz[*a*]anthracene, benzo[*b*]fluoranthene, benzo[*j*]fluoranthene, benzo[*k*]fluoranthene, chrysene, cyclopenta[*cd*]pyrene, dibenz[*a,h*]anthracene, dibenzo[*a,e*]fluoranthene, dibenzo[*a,e*]pyrene, dibenzo[*a,h*]pyrene, dibenzo[*a,i*]pyrene, dibenzo[*a,l*]pyrene, indeno[*1,2,3-cd*]pyrene) have been classified by EPA as probable human carcinogens (B2) (IRIS, 1992). Evidence for the carcinogenicity of PAHs in humans comes primarily from epidemiologic studies that have shown an increased mortality due to lung cancer in humans exposed to PAH-containing coke oven emissions, roof-tar emissions, and cigarette smoke (U.S. DHHS, 1990).

PAHs are ubiquitous in the environment and usually occur as complex mixtures with other toxic chemicals. They are components of crude and refined petroleum products and of coal. They are also produced by the incomplete combustion of organic materials. Many domestic and industrial activities involve pyrosynthesis of PAHs, which may be released into the environment in airborne particulates or in solid (ash) or liquid byproducts of the pyrolytic process. Domestic activities that

produce PAHs include cigarette smoking, home heating with wood or fossil fuels, waste incineration, broiling and smoking foods, and use of internal combustion engines. Industrial activities that produce PAHs include coal coking; production of carbon blacks, creosote, and coal tar; petroleum refining; synfuel production from coal; and use of Soderberg electrodes in aluminum smelters and ferrosilicum and iron works (Neff, 1985). Historic coal gasification sites have also been identified as significant sources of PAH contamination (J. Hesse, Michigan Department of Public Health, personal communication, March 1991).

Major sources of PAHs found in marine and fresh waters include biosynthesis (restricted to anoxic sediments), spillage and seepage of fossil fuels, discharge of domestic and industrial wastes, atmospheric deposition, and runoff (Neff, 1985). Urban stormwater runoff contains PAHs from leaching of asphalt roads, wearing of tires, deposition from automobile exhaust, and oiling of roadsides and unpaved roadways with crankcase oil (MacKenzie and Hunter, 1979). Solid PAH-containing residues from activated sludge treatment facilities have been disposed of in landfills or in the ocean (ocean dumping was banned in 1989). Although liquid domestic sewage contains <1 µg/L total PAH, the total PAH content of industrial sewage is 5 to 15 µg/L (Borneff and Kunte, 1965) and that of sewage sludge is 1 to 30 mg/kg (Grimmer et al., 1978; Nicholls et al., 1979).

In most cases, there is a direct relationship between PAH concentrations in river water and the degree of industrialization and human activity in the surrounding watersheds. Rivers flowing through heavily industrialized areas may contain 1 to 5 ppb total PAH, compared to unpolluted river water, ground water, or seawater that usually contains less than 0.1 ppb PAH (Neff, 1979).

PAHs can accumulate in aquatic organisms from water, sediments, and food. BCFs of PAHs in fish and crustaceans have frequently been reported to be in the range of 100 to 2,000 (Eisler, 1987). In general, bioconcentration was greater for the higher molecular weight PAHs than for the lower molecular weight PAHs. Biotransformation by the mixed function oxidase system in the fish liver can result in the formation of carcinogenic and mutagenic intermediates, and exposure to PAHs has been linked to the development of tumors in fish (Eisler, 1987). The ability of fish to metabolize PAHs probably explains why benzo[*a*]pyrene frequently is not detected or is found only at very low concentrations in fish from areas heavily contaminated with PAHs (Varanasi and Gmur, 1980, 1981).

Sediment-associated PAHs can be accumulated by bottom-dwelling invertebrates and fish (Eisler, 1987). For example, Great Lakes sediments containing elevated levels of PAHs were reported by Eadie et al. (1983) to be the source of the body burdens of the compounds in bottom-dwelling invertebrates. Similarly, Varanasi et al. (1985) found that benzo[*a*]pyrene was accumulated in fish, amphipod crustaceans, shrimp, and clams when estuarine sediment was the source of the compound. Approximate tissue-to-sediment ratios were 0.6 to 1.2 for amphipods, 0.1 for clams, and 0.05 for fish and shrimp. Although fish and most crustaceans evaluated to date have the mixed function oxidose system required for biotransformation of PAHs, some molluscs lack this system and are unable to metabolize PAHs efficiently (Varanasi et al., 1985). Thus, bivalves are good bioaccumulators of some PAHs. NAS (1991) reported that PAH contamination in bivalves has been found in all areas of the United States. Varanasi et al. (1985)

ranked benzo[*a*]pyrene metabolism by aquatic organisms as follows: fish > shrimp > amphipod crustaceans > clams. Half-lives for elimination of PAHs in fish ranged from less than 2 days to 9 days (Niimi, 1987). If PAHs are included as target analytes at a site, preference should be given to selection of a bivalve mollusc as one of the target species (if available) and a finfish as the other target species.

Three States (Massachusetts, Michigan, and Ohio) have issued advisories for PAH contamination in finfish (RTI, 1993).

Although several PAHs have been classified as probable human carcinogens (Group B2), benzo[*a*]pyrene is the only PAH for which an oral cancer slope factor (SF) is currently available in IRIS (1995). It is recommended that, in both screening and intensive studies, tissue samples be analyzed for benzo[*a*]pyrene, benz[*a*]anthracene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, chrysene, dibenz[*a,h*]anthracene, and indeno*[1,2,3-cd*]pyrene, and that the relative potencies given for these PAHs in the EPA provisional guidance for quantitative risk assessment of PAHs (U.S. EPA, 1993c) be used to calculate a potency equivalency concentration (PEC) for each sample for comparison with the recommended SV for benzo[*a*]pyrene (see Section 5.3.2.3). At this time, EPA's recommendation for risk assessment of PAHs (U.S. EPA, 1993c) is considered provisional because quantitative risk assessment data are not available for all PAHs. This approach is under Agency review and over the next year will be evaluated as new health effects benchmark values are developed. Therefore, the method provided in this guidance document is subject to change pending results of the Agency's reevaluation.

4.3.6 Polychlorinated Biphenyls (Total)

PCBs are base/neutral compounds that are formed by the direct chlorination of biphenyl. PCBs are closely related to many chlorinated hydrocarbon pesticides (e.g., DDT, dieldrin, and aldrin) in their chemical, physical, and toxicologic properties and in their widespread occurrence in the aquatic environment (Nimmo, 1985). There are 209 different PCB compounds, termed congeners, based on the possible chlorine substitution patterns. In the United States, mixtures of various PCB congeners were formulated for commercial use under the trade name Aroclor on the basis of their percent chlorine content. For example, a common PCB mixture, Aroclor 1254, has an average chlorine content of 54 percent by weight (Nimmo, 1985).

Unlike the organochlorine pesticides, PCBs were never intended to be released directly into the environment; most uses were in industrial systems. Important properties of PCBs for industrial applications include thermal stability, fire and oxidation resistance, and solubility in organic compounds (Hodges, 1977). PCBs were used as insulating fluids in electrical transformers and capacitors, as plasticizers, as lubricants, as fluids in vacuum pumps and compressors, and as heat transfer and hydraulic fluids (Hodges, 1977; Nimmo, 1985). Although use of PCBs as a dielectric fluid in transformers and capacitors was generally considered a closed-system application, the uses of PCBs, especially during the 1960s, were broadly expanded to many open systems where losses to the environment were likely. Heat transfer systems, hydraulic fluids in die cast machines, and uses in

specialty inks are examples of more open-ended applications that resulted in serious contamination in fish near industrial discharge points (Hesse, 1976).

Although PCBs were once used extensively by industry, their production and use in the United States were banned by the EPA in July 1979 (Miller, 1979). Prior to 1979, the disposal of PCBs and PCB-containing equipment was not subject to Federal regulation. Prior to regulation, of the approximately 1.25 billion pounds purchased by U.S. industry, 750 million pounds (60 percent) were still in use in capacitors and transformers, 55 million pounds (4 percent) had been destroyed by incineration or degraded in the environment, and over 450 million pounds (36 percent) were either in landfills or dumps or were available to biota via air, water, soil, and sediments (Durfee et al., 1976).

PCBs are extremely persistent in the environment and are bioaccumulated throughout the food chain (Eisler, 1986; Worthing, 1991). There is evidence that PCB health risks increase with increased chlorination because more highly chlorinated PCBs are retained more efficiently in fatty tissues (IRIS, 1992). However, individual PCB congeners have widely varying potencies for producing a variety of adverse biological effects including hepatotoxicity, developmental toxicity, immunotoxicity, neurotoxicity, and carcinogenicity. The non-ortho-substituted coplanar PCB congeners, and some of the mono-ortho-substituted congeners, have been shown to exhibit "dioxin-like" effects (Golub et al., 1991; Kimbrough and Jensen, 1989; McConnell, 1980; Poland and Knutson, 1982; Safe, 1985, 1990; Tilson et al., 1990; U.S. EPA 1993c). The neurotoxic effects of PCBs appear to be associated with some degree of ortho-chlorine substitution. There is increasing evidence that many of the toxic effects of PCBs result from alterations in hormonal function. However, because PCBs can act directly as hormonal agonists or antagonists, PCB mixtures may have complex interactive effects in biological systems (Korach et al., 1988; Safe et al., 1991; Shain et al., 1991; U.S. EPA, 1993c). Because of the lack of sufficient toxicologic data, EPA has not developed quantitative estimates of health risk for specific congeners. PCB mixtures have been classified as probable human carcinogens (Group B2) (IRIS, 1992; U.S. EPA, 1988a).

Of particular concern are several studies that have suggested that exposure to PCBs may be damaging to the health of fetuses and children (Fein et al., 1984; Jacobson et al., 1985, 1990). However, these studies are inconclusive due to a failure to assess confounding variables (J. Hesse, Michigan Department of Public Health, personal communication, 1992). In a more recent study of prenatal exposure to PCBs and reproductive outcome, birth size was found to be associated positively with PCB exposure, contrary to expectations (Dar et al., 1992). The results of these investigations clearly indicate the need for further study. Nevertheless, it may be appropriate for States in which PCBs are found to be a problem contaminant in fish or shellfish tissue to assess the need to issue consumption advisories, particularly for pregnant women, nursing mothers, and children.

PCBs have been included in eight major fish contaminant monitoring programs (Appendix C). A recent summary of the National Contaminant Biomonitoring Program data from 1976 through 1984 indicated a significant downward trend in total PCBs, although PCB residues in fish tissue remained widespread (Schmitt et al., 1990). Total PCBs were detected at 91 percent of 374 sites surveyed in the National Study of Chemical Residues in Fish (U.S. EPA, 1992c, 1992d). Currently, PCB

contamination in fish and shellfish has resulted in the issuance of consumption advisories in 31 States (Figure 4-3) (RTI, 1993).

PCBs may be analyzed quantitatively as Aroclor equivalents or as individual congeners. Historically, Aroclor analysis has been performed by most laboratories. This procedure can, however, result in significant error in determining total PCB concentrations (Schwartz et al., 1987) and in assessing the toxicologic significance of PCBs, because it is based on the assumption that distribution of PCB congeners in environmental samples and parent Aroclors is similar.

The distribution of PCB congeners in Aroclors is, in fact, altered considerably by physical, chemical, and biological processes after release into the environment, particularly when the process of biomagnification is involved (Norstrom, 1988; Oliver and Niimi, 1988; Smith et al., 1990). Recent aquatic environmental studies indicate that many of the most potent, dioxin-like PCB congeners are preferentially accumulated in higher organisms (Bryan et al., 1987; Kubiak et al., 1989; Oliver and Niimi, 1988). This preferential accumulation probably results in a significant increase in the total toxic potency of PCB residues as they move up the food chain. Consequently, the congener-specific analysis of PCBs is required for more accurate determination of total PCB concentrations and for more rigorous assessment of the toxicologic effects of PCBs.

Even though the large number of congeners of PCBs and their similar chemical and physical properties present serious analytical difficulties, analytical methods for the determination of PCB congeners have been improved in recent years so that it is now possible to determine essentially all PCB congeners in mixtures (Huckins et al., 1988; Kannan et al., 1989; MacLeod et al., 1985; Maack and Sonzogni, 1988; Mes and Weber, 1989; NOAA, 1989b; Smith et al., 1990; Tanabe et al., 1987). Both NOAA (MacLeod et al., 1985; NOAA, 1989b) and the EPA Narragansett Research Laboratory conduct PCB congener analyses and have adopted the same 18 PCB congeners for monitoring fish contamination. However, quantitation of individual PCB congeners is relatively time-consuming and expensive and many laboratories do not have the capability or expertise to perform such analyses. Some States currently conduct both congener and Aroclor analysis; however, most States routinely perform only Aroclor analysis.

For the purposes of screening tissue residues against potential levels of public health concern in fish and shellfish contaminant monitoring programs, the issue of whether to determine PCB concentrations as Aroclor equivalents or as individual congeners cannot be resolved entirely satisfactorily at this time, primarily because of a lack of toxicologic data for individual congeners.

Ideally, congener analysis should be conducted. However, at present, only an Aroclor-based quantitative risk estimate of carcinogenicity is available (IRIS, 1993) for developing SVs and risk assessment. Consequently, until adequate congener-specific toxicologic data are available to develop quantitative risk estimates for a variety of toxicologic endpoints, the EPA Office of Water recommends, as an interim measure, that PCBs be analyzed as Aroclor equivalents, with total PCB concentrations reported as the sum of Aroclors.

States are encouraged to develop the capability to perform PCB congener analysis. When congener analysis is conducted, the 18 congeners recommended by NOAA (shown in Table 4-3) should be analyzed and summed to determine a total PCB concentration according to the approach used by NOAA (1989b). States may wish to consider including additional congeners based on site-specific considerations. PCB congeners of potential environmental importance identified by McFarland and Clarke (1989) are listed in Table 4-3.

This interim recommendation is intended to (1) allow States flexibility in PCB analysis until reliable congener-specific quantitative risk estimates are available, and (2) encourage the continued development of a reliable database of PCB congener concentrations in fish and shellfish tissue in order to increase our understanding of the mechanisms of action and toxicities of these chemicals. The rationale for, and the uncertainties of, this recommended approach are discussed further in Section 5.3.2.3.

4.3.7 Dioxins and Dibenzofurans

Note: At this time, the EPA Office of Research and Development is reevaluating the potency of dioxins and dibenzofurans. Information provided below as well as information in Section 5.3.2.4 related to calculating toxicity equivalent concentrations (TECs) and SVs for dioxins/furans is subject to change pending the results of this reevaluation.

The polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are included as target analytes primarily because of the extreme potency of 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD). Extremely low doses of this isomer have been found to elicit a wide range of toxic responses in animals, including carcinogenicity, teratogenicity, fetotoxicity, reproductive dysfunction, and immunotoxicity (U.S. EPA, 1987d). This compound is the most potent animal carcinogen evaluated by EPA, and EPA has determined that there is sufficient evidence to conclude that 2,3,7,8-TCDD is a probable human carcinogen (B2) (IRIS, 1992). Concern over the health effects of 2,3,7,8-TCDD is increased because of its persistence in the environment and its high potential to bioaccumulate (U.S. EPA, 1987d).

Because dioxin/furan contamination is found almost exclusively in proximity to industrial sites (e.g., bleached kraft paper mills or facilities handling 2,4,5-trichlorophenoxyacetic acid [2,4,5-T], 2,4,5-trichlorophenol [2,4,5-TCP], and/or silvex) (U.S. EPA, 1987d), it is recommended that each State agency responsible for monitoring include these compounds as target analytes on a site-specific basis based on the presence of industrial sites and results of any environmental (water, sediment, soil, air) monitoring performed in areas adjacent to these sites. All States should maintain a current awareness of potential dioxin/furan contamination.

Fifteen dioxin and dibenzofuran congeners have been included in two major fish contaminant monitoring programs; however, one congener, 2,3,7,8-TCDD, has been included in seven national monitoring programs (Appendix C). Six dioxin congeners and nine dibenzofuran congeners were measured in fish tissue and shellfish samples in the National Study of Chemical Residues in Fish. The various dioxin congeners were detected at from 32 to 89 percent of the 388 sites surveyed, while

the furan congeners were detected at from 1 to 89 percent of the 388 sites surveyed (U.S. EPA, 1992c, 1992d). The dioxin/furan congeners detected at more than 50 percent of the sites are listed below:

- 1,2,3,4,6,7,8 HpCDD (89 percent)
- 2,3,7,8 TCDF (89 percent)
- 2,3,7,8 TCDD (70 percent)
- 1,2,3,6,7,8 HxCDD (69 percent)
- 2,3,4,7,8 PeCDF (64 percent)
- 1,2,3,4,6,7,8 HpCDF (54 percent)
- 1,2,3,7,8 PeCDD (54 percent).

Currently, 22 States have issued fish consumption advisories for dioxins/furans (Figure 4-4) (RTI, 1993).

Dioxins/furans should be considered for analysis primarily at sites of pulp and paper mills using a chlorine bleaching process and at industrial sites where the following organic compounds have been or are currently formulated: herbicides (containing 2,4,5-trichlorophenoxy acids and 2,4,5-trichlorophenol), hexachlorophene, pentachlorophenol, and PCBs (U.S. EPA, 1987d). If resources permit, it is recommended that the 17 2,3,7,8-substituted tetra- through octa-chlorinated dioxin and dibenzofuran congeners shown in Table 4-4 be included as target analytes. At a minimum, 2,3,7,8-TCDD and 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF) should be determined.

4.4 TARGET ANALYTES UNDER EVALUATION

At present, the EPA Office of Water is evaluating one metal (lead) for possible inclusion as a recommended target analyte in State fish and shellfish contaminant monitoring programs. A toxicologic profile for this metal and the status of the evaluation are provided in this section. Other contaminants will be evaluated and may be recommended as target analytes as additional toxicologic data become available.

Note: Any time a State independently deems that the analyte currently under evaluation and/or other contaminants are of public health concern within its jurisdiction, the State should include these contaminants in its fish and shellfish contaminant monitoring program.

4.4.1 Lead

Lead is derived primarily from the mining and processing of limestone and dolomite deposits, which are often sources of lead, zinc, and copper (May and McKinney, 1981). It is also found as a minor component of coal. Historically, lead has had a number of industrial uses, including use in paints, in solder used in plumbing and food cans, and as a gasoline additive. As recently as the mid-1980s, the primary source of lead in the environment was the combustion of gasoline; however, use of lead in U.S. gasoline has fallen sharply in recent years. At present, lead is used primarily in batteries, electric cable coverings, some exterior paints, ammunition, and sound barriers. Currently, the major points of entry of lead into the environment are

from mining and smelting operations, from fly ash resulting from coal combustion, and from the leachates of landfills (May and McKinney, 1981).

Lead has been included in six national monitoring programs (Appendix C). Lead has been shown to bioaccumulate, with the organic forms, such as tetraethyl lead, appearing to have the greatest potential for bioaccumulation in fish tissues. High concentrations of lead have been found in marine bivalves and finfish from both estuarine and marine waters (NOAA, 1987, 1989a). Lead concentrations in freshwater fish declined significantly from a geometric mean concentration of 0.28 ppm in 1976 to 0.11 ppm in 1984. This trend has been attributed primarily to reductions in the lead content of U.S. gasoline (Schmitt and Brumbaugh, 1990). Currently three States (Massachusetts, Missouri, and Tennessee) and American Samoa have issued fish advisories for lead contamination (RTI, 1993).

Lead is particularly toxic to children and fetuses. Subtle neurobehavioral effects (e.g., fine motor dysfunction, impaired concept formation, and altered behavior profile) occur in children exposed to lead at concentrations that do not result in clinical encephalopathy (ATSDR, 1988). A great deal of information on the health effects of lead has been obtained through decades of medical observation and scientific research. This information has been assessed in the development of air and water quality criteria by the Agency's Office of Health and Environmental Assessment (OHEA) in support of regulatory decisionmaking by the Office of Air Quality Planning and Standards (OAQPS) and by the Office of Drinking Water (ODW). By comparison to most other environmental toxicants, the degree of uncertainty about the health effects of lead is quite low. It appears that some of these effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold. The Agency's Reference Dose (RfD) Work Group discussed inorganic lead (and lead compounds) in 1985 and considered it inappropriate to develop an RfD for inorganic lead (IRIS, 1993). Lead and its inorganic compounds have been classified as probable human carcinogens (B2) by EPA (IRIS, 1992). However, at this time, a quantitative estimate of carcinogenic risk from oral exposure is not available (IRIS, 1993).

Because of the lack of quantitative health risk assessment information for oral exposure to inorganic lead, the EPA Office of Water has not included lead as a recommended target analyte in fish and shellfish contaminant monitoring programs at this time. **Note:** Because of the observation of virtually no-threshold neurobehavioral developmental effects of lead in children, States should include lead as a target analyte in fish and shellfish contaminant programs if there is any evidence that this metal may be present at detectable levels in fish or shellfish tissue. Additional information is provided on this issue in Volume II—Risk Assessment and Fish Consumption Limits—in this guidance series (U.S. EPA, 1994).